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Economic Evaluation of the effect of Quitting Smoking on Weight Gains: Evidence from the United Kingdom

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ABSTRACT

Objective: This article estimated the causal effect of quitting smoking on body weight gains in the United Kingdom to evaluate whether savings in health costs deriving from smoking prevention and its related diseases are greater than the costs associated with increased obesity. **Methods:** We used a longitudinal data set extracted from two waves (2004–2006) of the British Household Panel Survey, which includes information on smoking and a large number of sociodemographic variables. We modeled the effect of quitting smoking on body weight accounting for heterogeneous responses from individuals belonging to different clinical classes of body mass index (BMI) (i.e., overweight and obese individuals). National Health Service costs associated with smoking were then used to implement a cost-benefit analysis, comparing the advantages of smoking reductions with the costs associated with increased obesity. **Results:** The BMI was found to increase by 0.26 points for quitters compared with those who continued to smoke. The estimated BMI increase was larger for overweight (0.49 points) and obese (0.76 points) people. This result does not change when different control groups are examined. From an economic perspective, the National Health Service cost reductions attributable to quitting smoking were £156.81 million whereas the lost benefit for unintended increases in body weight was £24.07 million. **Conclusions:** This article found that the health benefits associated with quitting smoking are greater than the costs associated with increased overweight and obesity.

Keywords: Obesity, quitting smoking, BHPS dataset, health care costs and benefits.

Introduction

In the last few decades, obesity has become a substantial risk factor for a number of severe and chronic diseases that constitute the main causes of death, including heart disease, strokes, some types of cancer, and other serious life-shortening conditions such as type 2 diabetes. Similar patterns of the prevalence of overweight and obesity are shown in the United States and Europe, although in the old continent they reach a lower absolute level [1].

In the United Kingdom, obesity has constantly risen by 8 to 9 percentage points over the last 15 years, sex trends being similar. The burden on the National Health Service (NHS) associated with the excess weight was estimated to have increased in the period 1998 to 2006 from 1.5% to 2.6% of total health expenditure. Estimates by the NHS forecast that the cost to the service, directly attributable to obesity, may rise to £5.3 billion by 2025.

Over the last two decades, another clearly evident trend that has pervaded Western countries has been the decline in the rate of smoking. Simultaneous examination of smoking and body weight trends has led to mixed evidence on this relationship [2–5], although some recent works have established the existence of a significant negative causal nexus between smoking and body weight [6–10]. This result is also supported by the medical literature, which shows how smoking reductions imply changes in metabolic rates and eating habits, leading to the unintended consequence of weight gains [11–13]. These studies, however, never analyzed whether the savings in health care costs associated with quitting smoking were larger or smaller than the increased costs required in treating obesity.

In this article, we sought to contribute to this literature by comparing social costs due to increased obesity with benefits from quitting smoking in the United Kingdom. To achieve this aim, we evaluated the heterogeneous effects of quitting smoking for individuals belonging to various body mass index (BMI) clinical classes. We used a longitudinal data set extracted from two waves (2004–2006) of the British Household Panel Survey (BHPS) that includes information on smoking and a large number of sociodemographic variables. We exploited the fact that we observed a random sample of the population of smokers in two periods, in which some subjects made the transition from smoking to nonsmoking status. Our model allowed us to include

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a full set of interactions between treatment and BMI classes. In this way, we could estimate the effect of changes in smoking habits on BMI for overweight and obese individuals, which are of particular interest for policymakers.

Our empirical strategy used a *difference-in-differences* (DID) approach to control for time-invariant unobservable confounders and used various "control groups," in addition to the natural control group of "smokers," to account for other possible sources of bias related to reverse causality or omitted variables.

The article is organized as follows. First, we document a significant increase in body weight for quitters. Although point estimates are not very large in magnitude, weight is found to increase particularly in obese individuals. Second, sensitivity analysis generally confirms these findings when various control groups are used to account for various sources of bias. Third, results from a cost-benefit analysis indicate that quitting smoking implies much larger savings in health costs than the costs required in treating increased obesity.

Estimates of Health Care Costs Generated by Smoking and Obesity in the United Kingdom

There are very many works estimating the NHS costs of obesity and smoking in the United Kingdom, and so choosing which source to adopt to obtain reliable estimates is more difficult. Concerning the economic costs of smoking-related ill health in the United Kingdom, we follow the systematic review by Allender et al. [14]. The authors compared studies published between 1997 and 2007 and calculated the burden of ill health due to smoking in each country of the United Kingdom. In particular, population-attributable fractions for smoking-related diseases from the World Health Organization's Global Burden of Disease Project were applied to NHS cost data to estimate direct financial costs. After analyzing more than 4000 articles, the above authors concluded that 109,164 deaths were attributable to smoking-related disease in 2006, which were responsible for £5.17 billion in health care costs (5.5% of total health care costs).

In our assessment of weight excess in the United Kingdom, we preferred to use the study by The House of Commons Health Select Committee [15]. That study estimated that the total cost attributable to obesity (i.e., for individuals with a BMI of >30) was about £3340 million to £3724 million in 2002. About 30% of total costs were due to the direct health care costs of treating obesity and its consequences, including general practitioner consultations, in-patient and day case admissions, outpatient attendance, and the cost of drugs. The costs of treating obesity and its consequences were 2.3% to 2.6% of the NHS expenditure. The greater part of these costs, however, was attributable to treating the consequences of obesity, rather than obesity itself, including cardiovascular diseases, type II diabetes, stroke, angina, osteoporosis, and various types of cancers. There were also obesity-related costs generated by lost earnings (i.e., lost potential national output), which could be directly attributed to obesity. These were reported by McCormick and Stone [16] to be in the range of £2350 million to £2600 million, of which about 50% were attributable to premature mortality due to obesity and the other 50% to consequences of certified diseases related to obesity.

In this article, we used only direct costs to compare social costs due to increased obesity, with benefits from quitting smoking, because we did not have information about smoking costs generated by lost earnings and also because, as suggested by Morris [17], estimates of indirect costs connected with lost earnings are largely underestimated.

Methods

Data

The data set used in this work were extracted from the multipurpose BHPS, which reports information at both household and individual levels for a representative sample of the UK population. The original sample was composed of 5,500 households and 10,300 individuals, drawn from 250 areas of England, and was subsequently enlarged to include Scotland and Wales in 1999 and Northern Ireland in 2002. The data set has 18 waves: the first survey was conducted in 1980, but, for our purposes, we used a sample of two waves, the 14th and 16th waves, conducted, respectively, in 2004 and 2006 because data on height and weight were also collected. Although these two anthropomorphic characteristics were self-reported, the potential measurement errors over time are limited by the reduced time span covered by our sample (see, e.g., Shiely et al. [18]). We selected a balanced panel of 13,320 individuals for whom we had information about smoking habits and height and weight, which allowed us to calculate their BMI. Attrition is unlikely to be a problem in our data because the number of individuals who dropped out between the above two waves was quite limited. (The original sample was composed of 26,640 individuals, a number that later fell to 26,469. We also tested for differences between covariate distributions before and after balancing, and these were found not to be relevant. The tables are available from the authors upon request.) In fact, because nonresponse rates between 2004 and 2006 were very low, attrition problems were not likely to arise.

Model Structure

We examined a benchmark model in which BMI_{it} is the continuous measure of the BMI of an individual i at time t, and in which some fraction of the population reduces its cigarette consumption (e.g., nonrandom treatment). That is, individuals were observed in the pretreatment period $t=0$ and in the posttreatment period $t=1$, during which $D_{it}=1$ if an individual was exposed to the treatment between $t=0$ and $t=1$ and $D_{it}=0$ if not (control group).

From a theoretical point of view, we assumed that subjects "treated" in $t=1$ decided to reduce their smoking up to the extreme case of "zero cigarettes smoked" (i.e., quitting), a situation that is of great interest in the health economics literature [19,20]. With these premises, estimation of the causal effect of smoking on BMI was hindered by the presence of indigeneity, due to unobservable characteristics or reverse causality. To solve this problem, part of the literature uses a DID strategy with panel data (see, e.g., Baum [6] and French et al. [21]), which accounts for individual unobservable time-invariant characteristics affecting cigarette consumption and weight differently for treatment and control groups.

In view of the panel nature of our data set, we also adopted the DID approach and controlled for indigeneity by defining different control groups, to take account of the bias induced by reverse causality or time-varying unobservable characteristics (for more details, see the next section). We assumed that the outcome of interest (i.e., BMI) was generated through a component of variance process. A sufficient condition to identify the effect of smoking status changes is that selection for treatment, conditional on covariates, does not depend on individual transitory shocks. Because overweight and obese individuals are of great interest to policymakers, because preventing weight excess produces both significant gains in terms of health and reductions in terms of costs for treating their related illnesses, we considered possible heterogeneous treatment effects across BMI clinical thresholds. Provided that D is not endogenous, the standard DID specification is expressed by the following equation:

$$\mathrm{BMI}_{it} = \mu + \sum_{h=1}^{4} \eta_h (D \times B)_{ih} + \sum_{h=1}^{4} \delta_h (T \times B)_{th} + \sum_{h=1}^{4} \beta_h (D \times T \times B)_{ith} + \sum_{j=1}^{J} \pi_j X_{jit} + \varepsilon_{i,t} \quad (1)$$

where B_h, with h ranging from 1 to 4, is a categorical variable representing individuals classified as underweight (i.e., BMI $<$ 19), normal weight (i.e., BMI $\geq$ 19 and BMI $<$ 25), overweight (i.e., BMI $\geq$ 25 and BMI $<$ 30), or obese (i.e., BMI $\geq$ 30) in 2004. D_i is a dummy variable indicating treatment status for each individual i. We define quitters as treated, whereas control groups are discussed in next sections. T_t is a time dummy variable that indicates data collected in 2004 and 2006, respectively. The coefficient η_h associated with D_i captures any preexisting difference among treatment and control groups and for each BMI class; the coefficient δ_h associated with T_t is a proxy for unobserved variables that may affect BMIs of treatment and control groups. The heterogeneous effect of quitting across BMI classes is captured by β_h, and estimated as the interaction between D_i, T_t, and B_h, which represents the DID estimate of the average treatment effect (ATE) for group h in the two observed periods:

$$\beta_h^{\mathrm{DID}} = \mathrm{E}[\mathrm{BMI}_{01}^{h}|X] - \mathrm{E}[\mathrm{BMI}_{00}^{h}|X] - \mathrm{E}[\mathrm{BMI}_{11}^{h}|X] - \mathrm{E}[\mathrm{BMI}_{10}^{h}|X], \quad (2)$$

where for the sake of simplicity we indicate with BMI_{dt}^{h} the BMI of individuals with treatment status d interviewed at time t and for clinical threshold h.

Identification of Average Treatment Effects

We start our discussion on model identification by defining the following treatment and control groups.

D1. Treated group of smokers who quit smoking (TGQ): individuals who were smokers in 2004 and became nonsmokers in 2006.

D2. Control group of smokers (CGS): individuals who were smokers in 2004 and remained so in 2006.

In our model, quitters (i.e., treated individuals) were defined by a question asked in the BHPS about smoking status. The specific question is posed as "Do you smoke cigarettes?" and was recorded as a dichotomous variable. (The question posed in this way represents a limitation for our study, because it does not provide information about the frequency of smoking. Smoking and quitting smoking are chaotic unstable processes, and many attempts at quitting, e.g., are unplanned or spontaneous and many fail almost immediately, as suggested by West [22].) The BHPS also contains another question measuring smoking habits, which provides information about how many cigarettes the respondent usually smokes per day. In this article, we decided to use only the first question, about smoking participation, because we were more interested in the effects of quitting, rather than of smoking reduction. In order not to include subjects with unusual smoking behaviors in treatment or control groups, we used BHPS waves from 1999 to 2003 to reconstruct individual smoking histories. We then excluded from the sample those individuals who stated they were nonsmokers between 2004 and 2006, but who had been smokers between 1999 and 2003, and also those who stated they had been smokers between 1999 and 2003 but who were nonsmokers between 2004 and 2006.

Our evaluation strategy assumes that weight variations between 2004 and 2006 for TGQ individuals were affected by quitting smoking and by a spontaneous dynamic (i.e., time-specific component), whereas individuals who continued to smoke (CGS) were affected only by the spontaneous dynamic.

According to Cawley et al. [23], however, smoking habits are influenced by body weight if smokers do not quit because they are afraid of putting on weight. In this case, estimates of the relationship between smoking and body weight are biased by reverse causality. In addition, comparing TGQ with CGS may produce biased estimates because the estimated weight variation for the CGS is biased downward and consequently the estimated ATEs are biased upward.

We evaluated the magnitude of this effect by defining two control groups. The first also comprised nonsmokers in the smokers' control group (from this control group we excluded nonsmokers who started smoking in 2006, irrespective of whether they were or were not ex-smokers in 2004. We anticipate that the dimension within our sample [2.04%] is negligible for our estimates), that is, control group of smokers and nonsmokers (CGALL), whereas the second comprised only nonsmokers, that is, control group of nonsmokers (CGNS). Both groups were composed of individuals who kept their cigarette consumption stable and who, in principle, were not affected by reverse causality, like the CGS, because their weight did not affect their smoking decisions. We formally defined:

D3. CGALL: individuals who were smokers or nonsmokers in 2004 and remained so in 2006.

D4. CGNS: individuals who were nonsmokers in 2004 and remained so in 2006.

Because treatment was not randomly assigned in our data set, treated and control groups also differed according to time-varying unobserved factors related to smoking and weight decisions. In this case, the presence of unobserved heterogeneity may have caused our baseline estimates, comparing treatment groups (i.e., TGQ) with CGS, to be biased downward. In fact, quitters turned out to be generally more concerned about their health and more oriented toward the future, as discussed by McCaul et al. [24], and their decision to quit smoking may thus be seen as part of a more general attitude aimed at improving health—for example, also by reducing weight in obese people. The presence of these individuals in TGQ may have biased downward not only the estimated weight variation but also the estimated ATEs.

To take this aspect into consideration, we made up a new control group composed of individuals who were smokers in 2004 and 2006, but who quit in 2008. That is, the BMI variation of "next period" quitters was considered as the most appropriate control group for the BMI variation of TGQ because they had the most similar unobservable characteristics according to their future health behavior. A similar strategy was proposed and applied to the job market by Del Bono and Vuri [25]. Formally, our new control group was defined as follows:

D5. Control group of "next period" quitters (CG08): individuals who were smokers in 2004 and 2006, but who quit in 2008. (We used BHPS waves from 1999 to 2008 to reconstruct individual smoking histories. We then excluded from the sample those individuals who stated they were nonsmokers between 2006 and 2008, but who were smokers between 1999 and 2005 and also those who stated they were smokers between 1999 and 2005 but who were nonsmokers between 2006 and 2008.)

To obtain estimates as precise as possible about the effects of quitting smoking on BMI, we exploited the BHPS panel structure. In particular, we eliminated from CGNS and CGALL individuals who were smokers between 1999 and 2004 and fell from being CGS (CGNS or CGALL) individuals who were smokers (never smokers) in 2004 and who changed their smoking status in 2005, but who stated they were smokers (never smokers) in 2006.

Covariates

We exploited the ample information available in the BHPS to take into account a full set of observable individual characteristics in our models, both time-invariant and time-variant. All time-variant variables were introduced into the models as variations between waves, as in French et al. [21]. However, we exploited the fact that for most variables, other than BMI, we also had information from other BHPS waves to calculate variations between t and $t - 1$ and between $t - 1$ and $t - 2$. (Observations of individuals with a BMI of >40 or <15 were dropped from our sample. Note that weight in the BHPS is recorded as pre-pregnancy weight, whenever relevant.) Matrix X of covariates is composed of time-invariant covariates: sex, ethnic group membership, age, net income, education, and country dummies. Then, we included a set of time-variant sociodemographic covariates, reporting in our models both their absolute level and variations between t and $t - 1$ and between $t - 1$ and $t - 2$, related to physical activity, length of sickness, work conditions, marital status, and drinking habits. (The frequency of drinking outside the home and the frequency of physical exercise are included as variations only between t and $t - 2$ because they are recorded only biannually in the BHPS.)

Last, a full set of time-variant variables related to health shocks, which previous literature has demonstrated affects quitting smoking (see, e.g., Clark and Etilé [26]), was included, reporting both absolute levels and variations between t and $t - 1$ and between $t - 1$ and $t - 2$, for self-assessed health, visits to general practitioner, visits to outpatient departments, serious accidents, and health problems. Time-variant covariates were used to control for the effect of various lifestyle shocks, mostly health-related, which may also have had an effect on the decision to quit smoking and also on weight. In addition, the dummy variables recording whether subjects were underweight, normal weight, overweight, or obese in 2004 and a full set of interactions with the time dummy were included to account for differing BMI dynamics. These covariates are included as proxies of health concerns or orientation toward the future of individuals with different initial conditions in terms of BMI. Summary statistics of covariates for the treated (TGQ) and control groups are listed in Table 1 and Appendix A.1 in Supplemental Materials found at http://dx.doi.org/10.1016/j.jval.2015.06.008.

Results

Preliminary Analysis

Table 2 presents an average BMI increase of 0.85 points for TGQ and a smaller variation for CGS (0.24 BMI points). The unconditional ATE, according to these two groups, is therefore estimated to be 0.61 BMI points. According to other control groups, the estimated ATE is about 0.6 BMI points, except for CG08, in which it is slightly larger. We also used the usual BMI clinical thresholds to calculate unconditional ATEs for underweight, normal-weight, overweight, and obese individuals. Table 3 shows that the effect of quitting smoking on BMI is greater for obese individuals (range 0.97–1.08 points), irrespective of the control group used.

Main Estimates

Table 4 lists ordinary least squares estimates in which smokers are assumed to be the control group (CGS). For the reference category (normal-weight individuals, $h = 2$), we estimated an effect of quitting smoking on BMI of 0.31 (standard error [SE] = 0.122) and this coefficient was found to increase for individuals in the higher BMI clinical categories. For overweight individuals ($h = 3$), quitting smoking produced an extra BMI increase, with respect to the effect for the reference category, of 0.48 points (SE = 0.214), so that the total BMI increase for these individuals was 0.79. When we interacted the treatment with the obese category, we also found a positive and significant coefficient of 0.75 (SE = 0.384), implying a total BMI variation of 1.06 points. Last, underweight people presented a nonsignificant coefficient, meaning that their BMI variation after quitting was not statistically different from that of normal-weight people.

When we examined the δ coefficients, we found a positive and significant variation of 0.36 (SE = 0.065) for normal-weight people. Overweight and obese individuals showed lower trends with respect to the reference category, –0.20 (SE = 0.079) and –0.71 (SE = 0.14), respectively. To calculate the trends for the two categories, the effects estimated through the interaction terms had to be added to the parameter of the reference category. That is, the BMI of overweight and obese individuals varied by 0.16 and –0.35 points, respectively, between 2004 and 2006, consistent with the results from descriptive statistics presented in Table 2.

Table 4 also lists the estimated parameters from other control groups. When CGNS and CGALL were used as alternative control groups, the resulting ATEs were lower, but not very distant from those obtained from our baseline model. Last, we also show the estimates with CG08 as the control group. Although the ATE was in line with our expectations in terms of magnitude, it was not statistically different from zero. The small size of the obese subsample when CG08 was used as the control group, however, does not allow us to make accurate inferences. (With CG08 as the control group, there were 78 obese individuals per year and 102 in CGS, for a total of 180 obese individuals in the sample used to estimate the effect of quitting on BMI.)

Social Costs and Benefits

Our estimates identified the robust negative causal effect of smoking on body weight, which was larger in magnitude for overweight and obese people. This result is of crucial importance in the health economic literature because policymakers are interested in understanding whether the benefits from savings deriving from reduced smoking are larger or smaller than the extra costs generated by increased obesity.

Panel A of Table 5 shows the NHS costs associated with smoking in 2006, estimated by Allender et al. [14] to be £5170 million for the United Kingdom in 2006. Because 22% of the UK population smoked in 2006 (60,587,600), we can calculate a per-capita cost of smoking of £388. In addition, using the estimated total direct cost attributable to obesity of £3532 million in 2002 [15,16] and considering that the obese represented 24% of the total UK population in 2006, we obtained a per-capita cost of obesity of £2441. (To make costs comparable, the costs attributable to obesity in 2002 were divided by the consumer price index excluding tobacco, at base year 2002, provided by the NHS official statistics on price index data. The costs associated with smoking had already been estimated in 2006 pounds.)

Panel B of Table 5 lists the benefits of reduced smoking and the costs of the resulting increased obesity. From the BHPS, we can estimate that 2.75% of the smokers who were also classified as obese in 2004 quit smoking in 2006, and so this percentage can be used to calculate that the direct social benefits, in terms of NHS cost reductions attributable to smoking, were £156.81 million. This figure was obtained by multiplying the number of obese individuals who quit smoking (i.e., 400,000, 2.75% of 14.54 million obese individuals in 2004) by the per-capita cost of smoking (£388).

We also calculated the estimated social costs associated with increased obesity, attributable to quitting smoking. They arise from two different sources: 1) the extra costs of having to treat even those individuals who were overweight in 2004 and who, because of smoking-related weight increases, became obese in 2006; 2) the “lost benefits” of having to treat those

Table 1 – Descriptive statistics of covariates.

Variable	TGQ		CGS		CGNS		CGALL		CG08	
	2004	2006	2004	2006	2004	2006	2004	2006	2004	2006
Time-invariant covariates										
Sex										
Male	0.48	0.49	0.45	0.46	0.45	0.46	0.45	0.46	0.43	0.43
Female	0.52	0.51	0.55	0.54	0.55	0.54	0.55	0.54	0.57	0.57
Age class (y)										
18–29	0.27	0.23	0.23	0.23	0.14	0.13	0.17	0.16	0.26	0.22
30–39	0.25	0.25	0.24	0.22	0.18	0.16	0.19	0.17	0.24	0.24
40–49	0.14	0.17	0.2	0.22	0.19	0.2	0.2	0.2	0.21	0.22
50–59	0.13	0.12	0.17	0.17	0.18	0.17	0.18	0.17	0.13	0.15
60+	0.21	0.23	0.16	0.17	0.31	0.34	0.27	0.29	0.15	0.17
Ethnic group										
White	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99
Other	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Education										
Degree	0.13	0.13	0.07	0.07	0.16	0.17	0.14	0.14	0.08	0.1
Diploma	0.25	0.27	0.24	0.27	0.29	0.3	0.27	0.29	0.29	0.31
A-level	0.11	0.1	0.11	0.11	0.12	0.11	0.12	0.11	0.13	0.12
O-level	0.19	0.17	0.2	0.18	0.16	0.14	0.17	0.15	0.16	0.15
Low educated	0.32	0.33	0.38	0.37	0.28	0.29	0.31	0.31	0.33	0.32
Net income										
First quintile	0.23	0.19	0.27	0.26	0.2	0.17	0.22	0.19	0.24	0.21
Second quintile	0.18	0.21	0.22	0.23	0.19	0.19	0.2	0.2	0.21	0.24
Third quintile	0.23	0.21	0.22	0.2	0.21	0.19	0.21	0.19	0.22	0.17
Fourth quintile	0.21	0.2	0.17	0.17	0.21	0.21	0.19	0.2	0.21	0.23
Fifth quintile	0.15	0.19	0.12	0.14	0.2	0.23	0.18	0.21	0.12	0.16
Country										
England	0.53	0.54	0.45	0.44	0.49	0.5	0.48	0.48	0.46	0.46
Wales	0.15	0.15	0.17	0.18	0.17	0.17	0.17	0.17	0.17	0.16
Scotland	0.17	0.16	0.19	0.19	0.17	0.17	0.17	0.18	0.19	0.2
Northern Ireland	0.15	0.15	0.18	0.19	0.17	0.16	0.17	0.17	0.18	0.18
Time-variant covariates										
Marital status										
Married	0.47	0.51	0.42	0.43	0.61	0.62	0.56	0.57	0.44	0.47
Separated	0.03	0.02	0.04	0.04	0.02	0.02	0.02	0.02	0.04	0.03
Divorced	0.1	0.1	0.13	0.13	0.07	0.07	0.08	0.09	0.1	0.13
Widowed	0.04	0.05	0.05	0.06	0.09	0.1	0.08	0.09	0.05	0.05
Never married	0.35	0.31	0.36	0.35	0.22	0.2	0.26	0.24	0.37	0.32
Variations on marital status										
Married in t	0.03	0.03	0.03	0.03	0.01	0.02	0.02	0.02	0.02	0.03
Married in $t-1$	0.03	0.03	0.02	0.02	0.01	0.02	0.02	0.02	0.02	0.03
Separated in t	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.01
Separated in $t-1$	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.01
Divorced in t	0.02	0.02	0.01	0.02	0.01	0.01	0.01	0.01	0.02	0.02
Divorced in $t-1$	0.02	0.02	0.01	0.02	0.01	0.01	0.01	0.01	0.02	0.02
Widowed in t	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Widowed in $t-1$	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Strenuous job										
Yes	0.04	0.06	0.05	0.05	0.04	0.04	0.04	0.04	0.07	0.06
No	0.96	0.94	0.95	0.95	0.96	0.96	0.96	0.96	0.93	0.94
Variations on job strenuousness										
From strenuous to nonstrenuous in t	0.04	0.05	0.05	0.04	0.03	0.04	0.04	0.04	0.05	0.02
From nonstrenuous to strenuous in t	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.02
From strenuous to nonstrenuous in $t-1$	0.06	0.06	0.05	0.04	0.04	0.04	0.04	0.04	0.05	0.03
From nonstrenuous to strenuous in $t-1$	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.01
Drinking habits										
At least once a week	0.36	0.28	0.35	0.34	0.24	0.23	0.27	0.26	0.35	0.33
Less than once a week	0.64	0.72	0.65	0.66	0.76	0.77	0.73	0.74	0.65	0.67

continued on next page

Table 1 – *continued.*

Variable	TGQ		CGS		CGNS		CGALL		CG08	
	2004	2006	2004	2006	2004	2006	2004	2006	2004	2006
Variation on drinking habits										
From less than once to once a week between t and $t-2$	0.11	0.07	0.11	0.09	0.11	0.06	0.09	0.07	0.14	0.07
From once to less than once a week between t and $t-2$	0.09	0.09	0.09	0.09	0.07	0.07	0.07	0.08	0.11	0.09
Physical activity										
At least once a week	0.50	0.51	0.51	0.52	0.54	0.56	0.52	0.54	0.50	0.51
Less than once a week	0.50	0.49	0.49	0.48	0.46	0.44	0.48	0.46	0.50	0.49
Variation on drinking habits										
From less than once to once a week between t and $t-2$	0.04	0.05	0.07	0.05	0.09	0.06	0.08	0.06	0.04	0.06
From once to less than once a week between t and $t-2$	0.03	0.06	0.04	0.06	0.05	0.03	0.04	0.03	0.03	0.05
Long-term sickness										
Yes	0.06	0.06	0.08	0.09	0.03	0.03	0.05	0.05	0.07	0.05
No	0.94	0.94	0.92	0.91	0.97	0.97	0.95	0.95	0.93	0.95
Variation on long-term sickness										
From non–long-term to long-term sick in t	0.08	0.07	0.06	0.08	0.02	0.06	0.04	0.06	0.06	0.05
From long-term to non–long-term sick in t	0.01	0.01	0.02	0.02	0.01	0.01	0.01	0.01	0.02	0.02
From non–long-term to long-term sick in $t-1$	0.07	0.04	0.06	0.05	0.07	0.03	0.05	0.04	0.05	0.03
From long-term to non–long-term sick in $t-1$	0.01	0.01	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.02
Working women in household										
Yes	0.91	0.77	0.9	0.78	0.91	0.69	0.91	0.72	0.91	0.82
No	0.09	0.23	0.1	0.22	0.09	0.31	0.09	0.28	0.09	0.18
Variation on number of working women in household										
Between t and $t-1$	–0.01	–0.14	–0.01	–0.11	–0.01	–0.22	–0.01	–0.19	–0.01	–0.09
Between $t-1$ and $t-2$	0	0	0	0	0	0	0	0	–0.01	0.01
Job hours (including overtime)										
Number	30.86	29.05	28.51	28.26	27.42	26.96	27.72	27.29	29.68	30.19
Variation on job hours (including overtime)										
Between t and $t-1$	–0.34	–0.77	0.08	–0.22	0.07	–0.24	0.07	–0.23	–0.53	–0.8
Between $t-1$ and $t-2$	0.64	–1.12	–0.02	0.15	–0.13	–0.32	–0.1	–0.2	–0.01	1.31

Note. A-levels and O-levels refer to the examinations for the General Certificate of Education offered by educational institutions in the United Kingdom and a few of the former British colonies. In particular, A-levels were the subsequent examinations for those who studied for a further 2 years after O-levels at the age of 16 y.

CGALL, control group of smokers and nonsmokers; CGNS, control group of nonsmokers; CGS, control group of smokers; CG08, control group of "next period" quitters; TGQ, treated groups of quitters.

Table 2 – BMI absolute variations and ATEs (2004–2006) by smoking status group.

Groups	No. of observations	BMI		Absolute variation	ATE
		2004	2006	(2004–2006)	(2004–2006)
TGQ	491	25.04	25.89	0.85	–
CGS	3261	25.11	25.36	0.25	0.6
CGALL	11924	25.76	26.04	0.28	0.57
CGNS	8663	26.02	26.24	0.22	0.63
CG08	385	24.6	24.78	0.18	0.67

ATE, average treatment effect; BMI, body mass index; CGALL, control group of smokers and nonsmokers; CGNS, control group of nonsmokers; CGS, control group of smokers; CG08, control group of "next period" quitters; TGQ, treated groups of quitters.

Table 3 – BMI absolute variations and ATEs (2004–2006) by smoking status group and BMI class.

BMI class	Absolute variation				ATE			
	<19	19–25	25–30	>30	<19	19–25	25–30	>30
TGQ	1.13	0.71	1	0.78	–	–	–	
CGS	0.35	0.41	0.2	–0.3	0.81	0.3	0.8	1.09
CGALL	0.38	0.42	0.21	–0.21	0.73	0.27	0.75	0.97
CGNS	0.44	0.41	0.24	–0.18	0.72	0.29	0.75	0.98
CG08	0.41	0.36	–0.01	–0.20	0.76	0.33	1.01	0.99

Note. BMI clinical classes: <19, underweight; 19–25 normal weight; 25–30, overweight; >30, obese.
ATE, XXX; BMI, body mass index; CGALL, control group of smokers and nonsmokers; CGNS, control group of nonsmokers; CGS, control group of smokers; CG08, control group of "next period" quitters; TGQ, treated groups of quitters.

individuals who were already obese in 2004 and who, because they stopped smoking, did not become overweight in 2006. From our empirical model, the BMI in 2006 is a linear function of two components, $\delta+\beta_1$, all other characteristics remaining constant. The estimates of Equation 1 (Table 3) show the effect of quitting smoking on BMI of overweight individuals in 2004, which is $\beta_1+\beta_{1,3}=0.75$. To exclude those individuals who would have become obese because of other factors, we must add to this value the estimated BMI growth trend, represented by $\delta+\delta_3$, of 0.21. In other words, our model predicts that overweight

Table 4 – Causal effect of quitting smoking on BMI by clinical classes and control group.

Parameter	CGS	CGNS	CGALL	CG08
η_1	–0.09	–0.30*	–0.23†	0.01
	(0.114)	(0.107)	(0.105)	(0.161)
$\eta_{1,1}$	0.14	–0.11	0.01	–0.26
	(0.278)	(0.266)	(0.260)	(0.358)
$\eta_{1,3}$	–0.15	0.02	–0.03	0.13
	(0.176)	(0.167)	(0.164)	(0.241)
$\eta_{1,4}$	–0.41	0.07	–0.04	0.75
	(0.417)	(0.394)	(0.391)	(0.512)
β_1	0.26‡	0.30†	0.29†	0.25
	(0.135)	(0.125)	(0.124)	(0.199)
$\beta_{1,1}$	0.44	0.39	0.43	0.48
	(0.484)	(0.475)	(0.472)	(0.536)
$\beta_{1,3}$	0.49†	0.43†	0.44†	0.73†
	(0.228)	(0.216)	(0.214)	(0.327)
$\beta_{1,4}$	0.76‡	0.65‡	0.66‡	0.13
	(0.396)	(0.355)	(0.357)	(0.629)
δ	0.42*	0.44*	0.42*	0.43*
	(0.053)	(0.029)	(0.026)	(0.151)
δ_1	–0.08	0.06	0.00	0.01
	(0.107)	(0.085)	(0.065)	(0.255)
δ_3	–0.21†	–0.17*	–0.18*	–0.45‡
	(0.084)	(0.042)	(0.038)	(0.243)
δ_4	–0.72*	–0.61*	–0.62*	–0.20
	(0.158)	(0.067)	(0.062)	(0.503)
Constant	22.93*	22.78*	22.80*	22.84*
	(0.228)	(0.136)	(0.118)	(0.468)
Observations	5,361	15,200	19,741	1,540
R^2	0.80	0.80	0.80	0.78
Adjusted R^2	0.80	0.80	0.80	0.77

Notes. h = 1, 2, 3, 4 represents BMI clinical classes of underweight, normal weight, overweight, and obesity. Normal weight is the reference modalit y. The estimated parameters are those described in Equation 1:

$$\mathrm{BMI}_{it}=\mu+\sum_{h=1}^{4}\eta_h(D\times B)_{ih}+\sum_{h=1}^{4}\delta_h(T\times B)_{th}+\sum_{h=1}^{4}\beta_h(D\times T\times B)_{ith}+\sum_{j=1}^{J}\pi_j X_{jit}+\varepsilon_{i,t} \quad (1)$$

Standard errors are given in parentheses.
BMI, body mass index; CGALL, control group of smokers and nonsmokers; CGNS, control group of nonsmokers; CGS, control group of smokers; CG08, control group of "next period" quitters; TGQ, treated groups of quitters.
* $P \le 0.01$.
† $P \le 0.05$.
‡ $P \le 0.1$.

Table 5 – Social costs and benefits of quitting smoking.

A.	
UK population	60.59 million
Percentage of smokers	0.22
Cost of smoking (£)	5170.5 million
Percentage of overweight people	0.38
Percentage of obese people	0.24
Cost of obesity (£)	3550.4 million
Per-capita cost of smoking (£)	387.91
Per-capita cost of obesity (£)	244.1
Social benefits of quitting smoking (£)	156.81 million
B.	
$(\delta + \delta_3) + (\beta_1 + \beta_{1,3})$, CGS	0.21 + 0.75
$(\delta + \delta_4) + (\beta_1 + \beta_{1,4})$, CGS	–0.30 + 1.02
Obese quitters between 2004 and 2006 (estimated by BHPS) (%)	0.0275
Percentage of overweight individuals with a BMI of >29.05 and <29.85	4.23
Percentage of obese individuals with a BMI of <30.35 and >30	1.69
Social costs of increased obesity: overweight with a BMI of >29.05 and <29.85 (£)	17.20 million
Social costs of increased obesity: obese with a BMI of <30.35 (£)	6.87 million
Net social benefits of quitting smoking (£)	132.74 million

Notes. All values were calculated in 2006 pounds divided by the corresponding consumer price index with base year in 2002, provided by NHS official statistics on price indices data.
BMI, body mass index; CGS, control group of smokers; BHPS, British Household Panel Survey; NHS, National Health Service.

smokers who quit smoking and had a BMI of more than 29.04 (but <29.84) in 2004 would become obese in 2006 (BMI of >30 in 2006) because of quitting. This quota of individuals represents an extra cost that the NHS would not have sustained in a scenario in which nobody quits.

In addition, again looking at Table 4, our estimates indicate a negative trend for obese people ($\delta+\delta_4=-0.30$), implying that those who had a BMI of more than 30 but lower than 30.30 in 2004 would become overweight in 2006 and would not represent a cost (at least in terms of obesity) for the NHS. But, if we look at the obese individuals who also quit smoking in 2006, the positive effect associated with quitting, $\beta_1+\beta_{1,4}=1.02$, clearly overcompensates the natural decreasing trend of BMI and keeps a significant number of individuals trapped in obesity. These people represent a lost benefit for the NHS in terms of savings due to reduced obesity. All the other individuals classified as obese in 2006 would have been obese in any case, and consequently are not considered a burden for the NHS.

Again, panel B of Table 5 presents the estimates of the costs described previously. The percentage of individuals with a BMI of more than 29.04 and less than 29.80 in 2004, estimated by the BHPS, was 4.23% of the total population (i.e., 2.56 million people). If we assume that 2.75% of the UK population (i.e., a sample of 70,481) quit smoking in 2006, we obtain an additional cost of £17.20 million (70,481 × £2,441) for the NHS. Moreover, the percentage of individuals with a BMI of more than 30 and less than 30.30 in 2004 was 1.69% (i.e., 1024 million people). Also, in this case, we assume that 2.75% (i.e., 28,159 people) quit smoking and estimate a lost benefit of £6.87 million (28,159 × £2,441). Last, the net benefit of £132.74 million is estimated by subtracting these costs from the total benefit.

Conclusions

In this study, our estimates support the finding that quitting smoking influences weight gains in the United Kingdom. Regressions that account for the usual clinical thresholds also reveal a greater sensitivity of quitting on people in obesity status. In other important differences, our estimates reveal that the coefficients between smoking and body weight in the control groups are close to each other, excluding any significant upward or downward estimation bias of this relationship.

These findings add new evidence to the debate on antismoking policies and related benefits to decrease the number of potential smokers in the United Kingdom. We show that the reduction in smokers has significant, although limited, effects on weight gains, which are slightly larger in overweight and obese people.

From the economic perspective, our results indicate that the substantial health benefits in stopping smoking do not play a role in increasing the cost of overweight and obesity. Prudent estimates from a social cost-benefit analysis suggest larger savings in health costs from quitting smoking than the costs associated with increased obesity.

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Supplemental Materials

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